perficial glands disappeared, then appeared oedema of the lower extremity, which yielded to Röntgenotherapy; then left-sided pleural effusion (Plate 25), gradually yielding to treatments over the mediastinum, then abdominal pain—treatments now were irregular and unsatisfactory. Patient developed fever, sweats and loss of weight and finally died, three and one-half years after beginning treatment. Autopsy showed no appreciably enlarged glands in neck, axilla, or mediastinum, but the mesenteric and retroperitoneal glands were very markedly enlarged.

Features of case: Clinical course very similar to Case IX. Marked improvement under Röntgenotherapy, while regularly administered. Insufficient treatment later. Long course of medical treatment (arsenic, etc.), before Röntgenotherapy was given.

A review of these cases emphasizes the following points:

1. The wide dissemination which had occurred in the majority of the cases before a correct diagnosis was made, or proper treatment instituted. In some, this delay is chargeable to the patient but in others it demonstrates a lack of diligence or knowledge on the part of the attending physician.

2. The frequency of early mediastinal involvement is striking.

3. A diagnosis based upon the blood findings was not possible in any of these cases. The examinations were made by various members of the staff, including Dr. Addis, Dr. Mehrtens and Dr. Barnett. In the majority of cases, the number of transitional and large mononeuclear cells combined did not exceed either relatively, or absolutely, the normal limits of transitionals alone. We, therefore, cannot make a diagnosis of Hodgkin's disease without the histological examination of an excised gland.

4. In no case was there an excerebration of the disease traceable to the removal of a test gland.

5. Vaccines were of no avail in one case; Benzol was tried in a second case, but without effect.

6. Radical removal of involved glands was followed by local recurrence, when insufficient Röntgenotherapy was given over the operative field.

7. Removal of foci of infection produced no evident alteration in the course of the disease.

8. In all cases so far treated, the superficial glands have practically disappeared under Röntgenotherapy and have caused no further concern during the course of the disease. There has, therefore, been no apparent reason or justification for radical removal of such gland groups.

9. Mediastinal and abdominal glands are not subject to surgical removal.

10. With proper treatment it is possible to cause absorption of these deep glands. How long such enlargement can be kept under control by deep Röntgenotherapy has still to be determined, as none of these cases has been satisfactorily treated throughout the entire course of the disease.

11. The close parallelism between certain cases of lymphosarcoma and Hodgkin's disease, both in clinical manifestations, response to treatment and histological picture, is very striking and suggestive.

In conclusion I may say that:

1st. The etiology of Hodgkin's disease is still unsettled.

2nd. The diphtheroid organism seems likely to prove a mere saphrophite.

3rd. There is good reason to consider Hodgkin's disease a neoplasm closely related to lymphosarcomata and endotheliomata.

4th. Efforts must be made for earlier diagnosis.

5th. Diagnosis must as yet rest upon the examination of an excised gland and not upon the examination of a blood smear.

6th. Treatment of early localized cases should consist of the radical removal of the involved glands and the removal of foci of irritation, together with thorough and persistent Röntgen treatment of the operative field and of all neighboring lymphatic areas, especially the mediastinum. Cure seems possible in these cases.

7th. In the more advanced cases with wide dissemination, the only treatment is Röntgenotherapy. If this is given thoroughly and persistently, remarkable temporary results may be obtained and it is remotely possible that cure might be achieved.

8th. In the advanced cases I have seen no indication for surgery other than the removal of foci of irritation.

9th. Vaccine therapy has been a failure and there is little reason to expect more from the immune serum.

10th. The technical details of Röntgenotherapy have been omitted from this discussion.

In conclusion I wish to express my appreciation to the members of the staff of the Stanford University Medical School for their co-operation in this work and especially to Miss G. Dunn, the technician in the Department of Röntgenotherapy.

HEADACHE RESULTING FROM PATHOLOGICAL INTRA-ORAL AND INTRA-NASAL CONDITIONS.*

By ADOLPH B. BAER, M. D., San Francisco, Cal.

One of the greatest, if not the greatest, source of satisfaction and pleasure which I have derived from the practice of this specialty, has been the relief of the large number of headaches, head pains and neuralgias which have resulted from conditions arising within this field, and the greatest sufferers and the most intense pains have usually been cured by the treatment of a comparatively simple condition. We know, of course, that one of the most difficult symptoms to account for and to treat satisfactorily is headache. We know also that the great majority of head pains are systemic in origin. If, however, a thorough general examination fails to account for the headache, the patient must always be examined by a head specialist. I have been surprised at the number of cases, patient sufferers for years, never examined by a specialist, who have been immediately relieved by the treatment of an intra-nasal or intra-oral condition.

In this paper, without any idea that I am tell-

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ing you anything which all of you do not know already, I shall merely attempt to review briefly the intra-nasal and intra-oral conditions which can be the cause of head pains.

Headaches of nasal origin are peculiar. They are usually not constant in severity. They are intensified by stooping, jarring, prolonged mental work and loss of sleep. They are apt to be unilateral, worse in the morning (usually disappearing towards noon), and are increased on lying down. They are present independent of eye strain, and relieving an eye condition or an eye muscle strain will have no effect upon the headache. Eye headaches will practically always be relieved by resting the eyes or by applying the proper eyeglass correction.

Of course, there are certain headaches which will have both a nasal and an orbital origin, and correcting one condition will relieve but not entirely cure the headache. Or we may accentuate an eye strain by over use and precipitate a reflex nasal disturbance, and to that extent contribute toward its causation. This is illustrated when proper nasal treatment will cure a headache, even in the presence of uncorrected ocular defects of relatively high degree.

In character and degree a nasal headache may range from an occasional neuralgia, to a sense of tightness at the root of the nose between the eyes; to a continuous dull ache; to a fulminating, throbbing pain, radiating to all the branches of the trigeminal nerve. I have seen cases simulating migraine with a history of the circular band pain of neurasthenia, and the pain of a driving nail being forced into the vault of the head, disappear by relieving an intra-nasal pressure between the septum and a lower turbinate.

The various intra-nasal conditions which can be the cause of headache, have been best summarized by Grunwald as follows:

(1) Swelling of the mucosa, causing pressure contact upon nerves.
(2) Negative pressure within the sinuses.
(3) Positive pressure by sinus suppuration.
(4) Ulceration of the mucosa, involving nerve endings and seen in atrophic rhinitis.
(5) Autointoxication by reabsorption of purulent secretions.
(6) Any condition which causes acute congestion of the cranial circulation.
(7) The spheno palatine ganglion syndrome.
(8) And most important of all, direct contact, or abnormal points of contact, between the septum, turbinates, uncinate process, bulla ethmoidalis and septum tuberculi.

We will frequently overlook one or the other of these various conditions if we make but a single examination. At least three examinations at different times and under different conditions must be made, before we can arrive at a satisfactory conclusion. The severity of the headache bears no relation to the pathological lesion and we can very easily overlook a minor and apparently unimportant nasal change, which may account for a severe and intense headache.

Swelling of the mucosa of the middle turbinate impinging against a relatively high deviation of the nasal septum, by disturbance of the local circulation or causing pressure upon the spheno-palatine ganglion will cause a symptom complex—i. e., nausea, vomiting, vertigo and intense headache, often described as migraine. The headache is usually bilateral because the thickened septum will usually obstruct and cause pressure in both nostrils, and in consequence, compress both ganglia or their nerve distributions at the same time.

Many headaches which begin at puberty and cease spontaneously in later life may be explained by the fact that the nasal mucosa is no longer erectile. And these headaches are very often inherited because the abnormal anatomical intranasal bone formation is also inherited. There will be the same form of septum deformity in every member of the same family.

In the great majority of cases the deviation may be of very slight extent. A slight abnormality will produce marked symptoms in one patient, while a most extreme deformity will be borne with no discomfort whatever by another. *High deviations, far back in the nose, are usually the most important.* The posterior part of the middle turbinate—i. e., the region over the sphenopalatine ganglion—seems to be the point of greatest irritation. The convergence of the bony walls above allows a relatively small deflection to cause a marked pressure and obstruction when the mucosa swells.

During the interval between the attacks, when the mucosa is normal, we may completely overlook the deviation. During the attack it is not difficult to demonstrate the deviation, the swelling and the resulting contact and compression. Many patients will complain of the nasal obstruction, and speak of the stuffiness of the head and nose, or refer to the fulness and pain in the nose between the eyes. But as a rule the pain will overshadow the subjective symptoms and in the interval between the attacks they will be too slight to attract the patient's attention. On the other hand, the obstruction is at times so marked and the contact point so sharp, that separation is impossible.

I believe that a thorough and complete submucous resection will cure most of these cases. But the operation must extend from the cribriform plate to palatine crest, and posteriorly to the anterior wall of the sphenoid. No amount of tinkering with the turbinates will do more than temporarily relieve the condition. A secondary hypertrophy of the removed turbinates, or a compensatory hypertrophy of one of the other turbinates, will soon restore the original pressure against the septum. After complete removal of all spines and spurrs and thickenings, of the septum tuberculi in front, and of the long spines which are so frequently seen posteriorly and high up, no possible amount of turbinate swelling will be able to cause pressure, for the resilient membranous partition will give before any turbinal overgrowth, should it occur.

It is not uncommon for patients to date the onset of a headache from a blow upon the nose, and not a few will refer the pain to the eyes. For these injuries will usually result in a high septal deformity, leaving the lower two-thirds of

the nose sufficiently clear to permit of perfectly free breathing and the condition will result most commonly in an eye headache; usually a dull ocular pain, which is increased when the patient uses the eyes. But correcting a possible eye condition which should, of course, always be done first, will have little or no effect, while a high septum operation will always clear up the headaches.

And there is another group, where the nose, unless examined during the stage of turgescence, will be regarded as normal. They belong to the neurotic group whose whole vasomotor system is unstable and toneless. Periodic headache in this group, caused by the increased turgescence of a turbinate, and consequent pressure upon a slightly deviated septum, will invariably be relieved by a turbinate operation, and they will be permanently cured by a septum operation.

We must not, however, in our zeal to correct septal deformities, overlook the lateral nasal wall. Our entire pathology may be caused by a prominent and hypertrophied uncinate process. This process may be so enlarged as to completely occlude the distal part of the nasal cavity. I have been surprised at the complete relief from headache which has reesulted from its removal.

The same may be said of prominent bulla ethmoidalis; the pressure of polyps from the frontal and ethmoid sinuses and the antrum; less frequently from hypertrophied posterior tips on the lower turbinate. And these lateral obstructions will occasionally be the cause of a sinus condition by causing the retention of normal secretions within the sinuses, with a resulting headache. It may be regarded as a prepurulent stage, but clinically there is no evidence of a purulent condition present. Nasal examination is negative, as are also X-ray examination and examination by suction. And the condition may last two, three or four months before the purulent stage is reached. But in the meantime the patient will complain of the most intense headache, which will be immediately and completely relieved by washing out the sinuses, usually, in fact, only the antrum.

I believe that it is impossible to associate within the nose, certain areas or regions with definite area, for referred pains about the head. No definite localization *even* for sinus headache is possible. The site of the pain helps very little in determining which sinus is affected. Only a thorough and complete examination of all the sinuses by the very exact methods now at our disposal will enable us to locate a headache within one or the other of the nasal sinuses.

Antrum pains are usually supra-orbital; less frequently infra-orbital; and they may radiate backward to the ear and occipital region.

Ethmoid and frontal headaches are usually referred to the ocular or frontal region. But a maxillary sinusitis may cause a reflex pain in the supra-orbital nerve similar in all respects to that caused by a frontal sinus infection. How often do we expect to find a frontal sinus infection which an examination turns out to be confined to the antrum.

Sphenoid sinus headache patients seem to be unable to describe or locate the pain. Most often it is a sort of boring pain toward the middle of the head far back between the eyes. But it may be parietal of occipital. And it may be located on the top of the head or radiating from the mastoid toward the top. In fact, occipito-parietal headache is always an indication for sphenoid sinus exploration.

Unfortunately, we occasionally get a frontal headache from involvement of the sphenoid, and we often get occipital headache in involvement of the frontal sinuses.

All of these sinus headaches may be caused by a simple catarrhal condition of the nasal mucosa with swelling and closure of the normal sinus openings. This results in a negative pressure or vacuum headache, which will be relieved by relieving the congestion and admitting a free supply of air. Or the condition may become chronic and persist even after the exciting cause has been removed.

Spheno-palatine ganglion irritation accounts for many intra-nasal headaches. It lies in the sphenomaxillary fossa, between the pterygoid process of the sphenoid and the superior maxilla; and is therefore in very close anatomical relationship to the sphenoid and posterior ethmoid sinuses.

It is therefore easy to understand how the ganglion may become infected directly from the nasal cavity (for it lies just below the surface of the nasal mucosa) or it may become involved secondarily by infection spreading from the sphenoid or ethmoid sinuses.

Sluder describes a symptom complex of sphenopalatine ganglion origin consisting of pain beginning at the root of the nose, extending to the upper jaw (and at times to the lower), backward under the zygoma and into the ear and mastoid, and thence to the neck, shoulder, arm and hand. When this condition is confined to the ganglion and is not secondary to a sphenoid or ethmoid sinus condition, there will usually be an inequality in the two sides of the soft palate; the arch on the affected side being higher, the uvula and median raphe being drawn to the opposite side. There is also a partial anesthesia and loss of taste on the affected side.

Naso-fibroma, and malignant growths in the nasal cavity and its sinuses, painful in themselves and by pressure, are fortunately rare.

In searching for a possible rhinological cause for headache, we will very often find the cause of trouble in the mouth. The ten years during which I practiced dentistry and oral surgery have proven of inestimable value to me in locating reflex or referred pains about the head, in pathological conditions occurring in the mouth and teeth.

These head pains or headaches may be located within the mouth itself, or they may spread reflexly over the head or be referred to other distant and remote parts of the body. The various tooth conditions causing such pains are: Dental caries; pulp stones; pyorrhea; chronic abscesses at the roots of apparently healthy teeth, with necrosis in the surrounding areas of the maxillary bones; acute abscesses; pressure by tooth roots, and by

unerupted teeth or impacted molars; supernumerary teeth; periostitis; osteomyelitis and maxillary bone necrosis; salivary calculi, cysts of sublingual and submaxillary glands.

And the most intense pains may be caused in perfectly normal mouths by an intra-nasal condition irritating reflexly the superior and inferior divisions of the fifth nerve.

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THE TREATMENT OF INFANTILE PARALYSIS.*

By JOHN CARLING, M. D., Los Angeles.

Before taking up the treatment of infantile paralysis, it might be well to refer briefly to the causes of deformity in this disease. They are: 1. Gravity. 2. The contraction of unopposed muscles. 3. Habitual posture. 4. Functional use.

To illustrate: If the dorsal flexors of the foot are paralyzed, the foot falls forward from the force of gravity plus contraction of the calf muscles. If the foot is not supported, structural shortening sets in and the limb becomes permanently deformed. If, however, the dorsal flexors are intact and the calf muscles are paralyzed the force of gravity is overcome by contraction of the dorsal flexors.

Habitual posture may cause deformity as in complete paralysis when the limbs are placed in certain positions for convenience.

In incomplete paralysis, when the patient begins to use his limbs further deformity is developed by the weight of the body and the attempt of the remaining muscles to do the work of those which are paralyzed.

Subluxation sometimes occurs from relaxation of ligaments around a joint and lack of muscular support, but complete dislocation is rare.

Deformities of the upper extremity are as a rule not so severe as those of the lower, because of the absence of strain due to weight bearing and the fact that gravity is opposed to muscular contraction.

Paralysis of the muscles of the shoulder may cause subluxation of the head of the humerus and paralysis of the muscles of the forearm, distortion of the hand from contraction accommodation and atrophy.

Paralysis of the trunk muscles may cause lateral curvature of the spine. The curvature in these cases is not towards the healthy side as might be supposed, but towards the paralyzed side. This is because the muscles of respiration are involved, producing a caving in of the chest on the affected side and the compensatory enlargement of the chest on the opposite side draws the spine towards it.

In infantile paralysis the extent of ultimate deformity is not limited to the muscles alone, but all the tissues of the affected part share in the atrophy and retardation of growth. It is evident that retardation of growth will be greater during the period of active development, consequently the younger the patient when attacked the greater will be the atrophy and shortening.

TREATMENT.

Active treatment of the paralyzed muscles should not begin until the acute inflammation in the cord has subsided. This may take from one to four weeks or longer and is indicated by the absence of pain and tenderness on handling the limbs. To overcome the tendency to deformity the joints should be manipulated several times daily and the limbs massaged to improve the circulation and nutrition of the muscles.

The galvanic current is of use in obtaining contraction of muscles that cannot be contracted voluntarily. When given alone, however, at irregular intervals it is of little value in restoring function and should therefore be always combined with massage and muscle training. The latter is by far the best means at our disposal for restoring lost power to the disabled muscles. It consists in aiding the patient to perform certain movements with the hope of stimulating impulses from the brain to the weakened or paralyzed muscles.

To illustrate a case: If the dorsal flexors of the foot are weakened and unable to act alone, the foot is dorsally flexed by the hand of the operator and the patient is directed to assist. If there is any response, and there usually is, less and less aid is given by the operator as the power returns. By patient and persistent efforts, muscles which are apparently hopelessly paralyzed, may be trained to perform their functions in whole or in part.

In infantile paralysis a hemorrhagic myelitis has attacked the cord and caused more or less destruction. Certain centers may have been completely destroyed while others may have escaped with only slight injury. Moreover some centers may have escaped injury altogether, but their associate centers having been destroyed and being unaccustomed to act alone, their function is lost unless trained to co-ordinate with other centers. Therefore, there exists in every disabled limb a certain amount of muscular power which is not evident and which cannot be made available unless cultivated. The patient, if a child, should never be left to do his own exercises, but should always be aided by parent or nurse. As the response of a muscle depends on the strength of the stimulus, the volition of the patient is greatly aided by a word of command. The exercises should be given daily under the direction of the physician and should be continued as long as improvement is noticeable. If contractions are present they should be overcome before the exercises are begun as it is impossible to strengthen a muscle until the strain on it has been relieved.

MECHANICAL TREATMENT.

The object of a brace is to prevent deformity due to contraction of the unopposed muscles and at the same time encourage functional use of the limb.

In paralysis of the extensor muscles of the leg, the foot drops forward and drags upon the ground in walking, causing the patient to awkardly lift the

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